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Reduced models of striatal neurons: dopamine modulation and dynamics

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Loss of dopamine cells in Parkinson's Disease and its animal models leads to profound motor deficits. An intact dopamine system also seems to be critical for many forms of learning [1]. Much work on understanding these roles of dopamine has focused on the striatum, the main input nucleus of the basal ganglia, as the striatum is the main locus of dopamine's action in the verterbrate brain [2]. Damage to the striatum itself also impairs both motor action and learning [3]. Thus, the twin problems of understanding the computational roles of dopamine and the striatum are inseparably intertwined.

Understanding dopamine's effects on the complex striatal microcircuit ideally requires large-scale models that replicate the neuron types, numbers, and connectivity at one-to-one scale. To build at such scales, we require individual neuron models that are simple enough to be computationally tractable, but sufficiently complex to capture key membrane properties that contribute to the characteristic behavior of a neuron species. Our neuron model of choice is the recent canonical spiking model of Izhikevich [4]. However, it has not yet been extended to account for the action of neuromodulators.

We extend the striatal medium spiny (MSN) and fast-spiking (FS) interneuron models of [5] to account for dopaminergic modulation of intrinsic ion channels and synaptic inputs. We use data from a recent 189 compartment model of the MSN [3] to tune our simple model of that neuron under both current injection and spiking input regimes with varying activation of dopamine D1- and D2-type receptors. The reduced models capture the input-output relationships for both current injection and spiking input with remarkable accuracy. We derive a full set of stability properties for the original and dopamine modulated forms of the MSN model. We use these to establish that the dopamine models do not change the stability properties and hence the models predict that the MSN is not bistable in either baseline or dopamine-saturated conditions. Our extensions to the simple model of the FS interneuron are consistent with the existing data, but tuning the new parameters is made difficult by the lack of quantitative results from experimental work. Our work thus establishes reduced yet accurate dopamine-modulated forms of MSN and FS interneuron models, suitable for use in large-scale models of the striatum. Moreover, these also provide a tractable framework for further study of dopamine's effects on individual neuron computation.

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